Case reports

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Ventricular septal defect and mitral regurgitation secondary to myocardial infarction
A case treated medically with long survival

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A patient is described who developed both a ventricular septal defect and mitral regurgitation after myocardial infarction. The severity of these was confirmed by cardiac catheterization. He was treated medically and survives in active good health 7 years later.

Both ventricular septal defects and mitral regurgitation are well-recognized complications of myocardial infarction. It is rare for both to occur in the same patient and the recent description of a case treated surgically (Rawlins, Mendel, and Braimbridge, 1972) has stimulated this report.

Case report

In 1965 a 60-year-old river board foreman was seen in his home. He gave a classical history of myocardial infarction. This had begun with 10 days of increasingly severe angina followed by prolonged chest pain 4 days previously. His transaminases were raised, and in leads II, III, and aVF of the electrocardiogram there were deep pathological Q waves, together with ST elevation and T wave inversion. In leads V5 and V6 there were also pathological Q waves with flat T waves.

He was admitted to Papworth Hospital and the blood pressure was 140/80 mmHg, and there were no abnormal physical signs. Three days after admission he had further chest pain of two hours' duration. His venous pressure had risen and there was a systolic murmur heard maximally in the fourth left intercostal space and also at the apex. It was at this time suggested that he had ruptured his ventricular septum. His condition remained good, but his heart failure was slow to respond to treatment, and 9 days after his admission his venous pressure still showed a 10 cm V wave. He remained in sinus rhythm throughout. It was now thought that he might have ruptured his ventricular septum and a papillary muscle. His progress from then on was uninterrupted. He was maintained on anticoagulants, digoxin, diuretics, and a low fat diet. He was discharged for convalescence, and readmitted 11 days later for cardiac catheterization, just over two months after his myocardial infarct. By that time he was feeling well, and had no more than slight exercise intolerance due to dyspnoea. The signs remained unchanged. The electrocardiogram showed only evolutionary abnormalities. The *x*-ray showed moderate cardiomegaly, and the lung fields looked definitely plethoric.

The salient features at cardiac catheterization are given in the Table.

TABLE

Site	O_2% *saturation*	*Pressure S/D mmHg*
Superior vena cava	59	
Right atrium		
High	59	a = 7
Low	56	x = −3
		v = 4
		y = 3
Inferior vena cava	74	
Right ventricle	87	
	95	
	89	74/−4 to 10
	90	
Pulmonary artery	81	70/10:40
LPCV		a = 20:27 (Fig. 1)
		x = 14
		v = 40
		y = 10
Right brachial artery	93	110/60
SBF = 3·7 l./min		
PBF = 10·7 l./min		(Fig. 2)
PVR = 1–2 units		

LPC = left pulmonary capillery venous; SBF = systemic blood flow; PBF = pulmonary blood flow; PVR = pulmonary vascular resistance

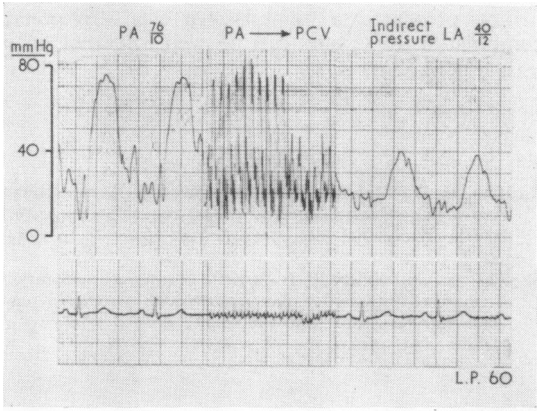

FIG. 1 *Pressure pulses recorded at right heart catheterization over two months after infarction. The pulmonary hypertension and the 40 mm V wave in the wedge trace are well shown.*

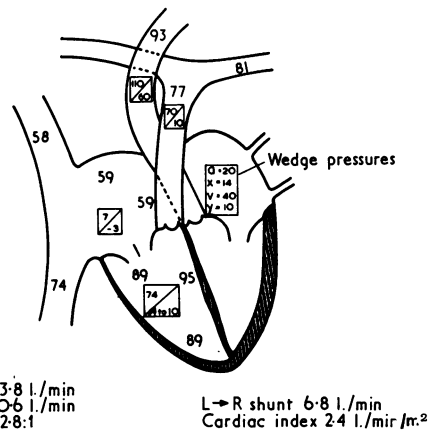

FIG. 2 *Diagrammatic representation of findings at cardiac catheterization. Pressures are enclosed in squares. The other figures are oxygen saturations.*

A dye curve was consistent with a considerable left-to-right shunt.

In view of the two haemodynamically severe lesions, and the patient's well-being, an operation was not advised and he was followed with great care and some apprehension. However, he has never looked back, and 7 years later there has been no regret about the policy adopted. He quickly returned to full work and after 6 months his anticoagulants were discontinued and he was started on clofibrate; his plasma cholesterol was 320 mg/100 ml. He was seen at increasing intervals. In 1969 (aged 64) he was reviewed in detail. His treatment continued. He was working full time, and his work

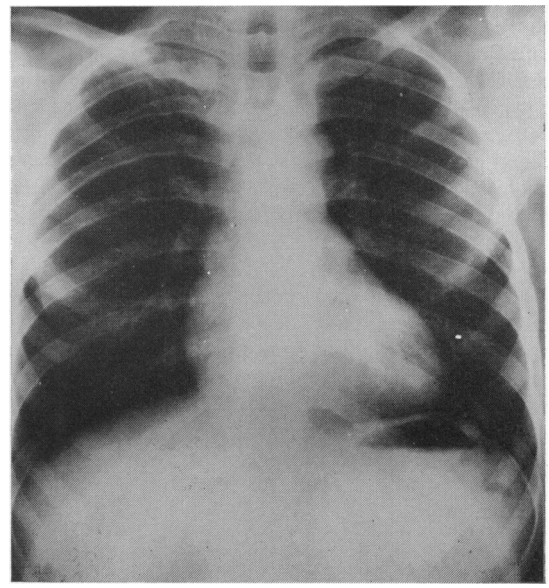

FIG. 3 *Chest x-ray in November 1971, over 6 years after the original episode. The moderate cardiomegaly and the pulmonary plethora have remained unchanged.*

involved a great deal of heavy walking on river banks and building-sites. He had absolutely no symptoms. The physical signs of both defects were still present. The electrocardiogram and chest *x*-ray showed no significant changes (Fig. 3). The findings on fluoroscopy were consistent with both defects still being present. The patient was not keen to be reinvestigated by cardiac catheterization, and there seemed no justification for pressing this.

At the age of 65 he retired, but was so fit, having no complaints, that he returned to his former work part time. In 1972, at the age of 67, he continues as before, and the findings are unchanged.

Discussion

There can be little doubt that this patient developed a ventricular septal defect and mitral regurgitation after myocardial infarction. The absence of the signs of these at the time of admission to hospital and the clear-cut episode with the appearance of the signs strongly suggest that one or both lesions were produced on the third day of his hospital stay (probably the seventh day after his infarction). Cardiac catheterization was carried out over two months after the infarction. The evidence of a large shunt through a ventricular septal defect, together with severe mitral incompetence, is unequivocal. If either lesion had been present on its own, it is likely that the patient would have been recommended for surgical treatment. It was felt that the presence of

these two lesions would greatly increase the mortality of operation, and it was therefore not suggested. The well-being of this patient over a period of 7 years has been remarkable. Although cardiac catheterization has not been repeated, all the indirect evidence suggests that both lesions have changed little. In any case, if an operation had been recommended in this patient the decision would have been made at about the time catheterization was carried out.

Rawlins *et al.* (1972) describe a similar case who died after operation. They emphasize the severity and the rarity of this combination of lesions after myocardial infarction. They review the published reports and have only been able to find one other case (Skoulas and Beier, 1967). This was a necropsy in a man aged 68 years.

This present case suggests that even this severe combination of lesions can be well tolerated in at least one patient.

References

Rawlins, M. D., Mendel, D., and Braimbridge, M. V. (1972). Ventricular septal defect and mitral regurgitation secondary to myocardial infarction. *British Heart Journal*, **34**, 322.

Skoulas, A., and Beier, R. L. (1967). Dissecting perforation of infarcted intraventricular septum with associated posterior papillary muscle involvement. *American Journal of Medicine*, **43**, 461.

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Notice

Third World and Ninth European Congress on Ballistocardiography and Cardiovascular Dynamics Sofia, 16–18 April 1973

The Third World and Ninth European Congress on Ballistocardiography and Cardiovascular Dynamics will take place in Sofia, Bulgaria, on 16, 17, and 18 April 1973.

The European Society for Ballistocardiography and Cardiovascular Dynamics holds its Congresses every second year, and every fourth year the European and American scientists in this field meet in a joint World Congress on Ballistocardiography and Cardiovascular Dynamics. The last World Congress was held in Oporto (Portugal) in 1969 and the last European Congress from 5 to 8 April 1971 in Ljubljana (Yugoslavia).

The principal themes of the present congress are: prognostic and diagnostic aspects of ballistocardiographic methods; and theoretical and technical aspects of ballistocardiographic methods. The noninvasive methods for investigation of cardiovascular dynamics will be discussed: ballistocardiography, seismocardiography, dynamocardiography, kinetocardiography, apex cardiography, plethysmography, and phonocardiography.

The meetings are interdisciplinary, and are of interest to cardiologists, physiologists, pathophysiologists, pharmacologists, biophysicists, and hydrodynamic, mechanical, and electrical engineers and other specialists in the technical and biomedical sciences. It is expected that 200 to 300 participants will attend the congress.

Applications as well as conditions for participation may be obtained from Dr. Alexander A. Talakov, Cardiovascular Centre, Experimental Sector, Miko Papo 65, Sofia 9 (Bulgaria).